

=> fil hcap
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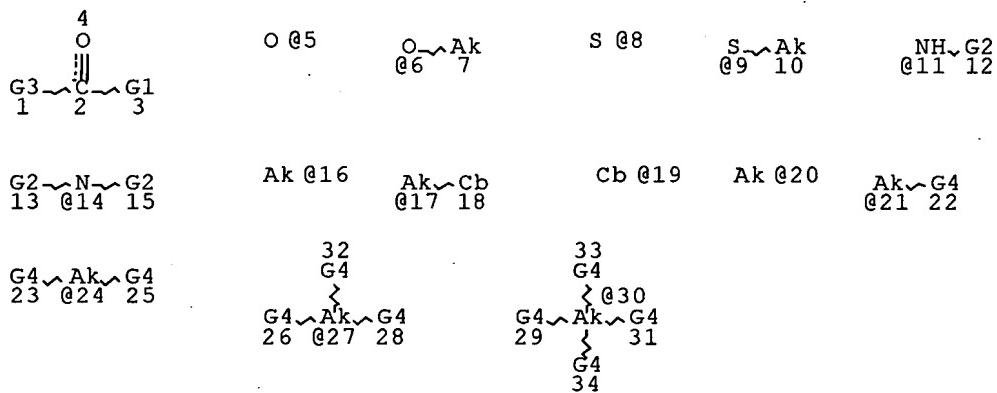
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FILE COVERS 1907 - 24 Apr 2007 VOL 146 ISS 18
FILE LAST UPDATED: 23 Apr 2007 (20070423/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

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L7      4887 SEA FILE=HCAPLUS ABB=ON   PLU=ON   COGNITION ENHANCERS+PFT/CT
L8      5895 SEA FILE=HCAPLUS ABB=ON   PLU=ON   COGNITION+PFT/CT
L9      12821 SEA FILE=HCAPLUS ABB=ON   PLU=ON   (L6 OR L7 OR L8)
L14     6530 SEA FILE=HCAPLUS ABB=ON   PLU=ON   L9 AND (PY<2003 OR PRY<2003
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ECOUNT	IS X30 C AT	30

GRAPH ATTRIBUTES:

RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 34

STEREO ATTRIBUTES: NONE

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L45	220 SEA FILE=HCAPLUS ABB=ON PLU=ON L42 AND L43

=> d 145 ibib ab hitind hitstr 1-10 100-120 200-220

L45 ANSWER 1 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2006:756033 HCAPLUS Full-text
 DOCUMENT NUMBER: 146:212690
 TITLE: Composition energizing and stimulating gonadal and cognitive activities
 INVENTOR(S): Scorei, Vilma; Scorei, Romulus
 PATENT ASSIGNEE(S): Rom.
 SOURCE: Rom., 3pp.
 CODEN: RUXXA3
 DOCUMENT TYPE: Patent
 LANGUAGE: Romanian
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
RO 120686	B1	20060630	RO 2001-783	20010706 <--
PRIORITY APPLN. INFO.:			RO 2001-783	20010706 <--

AB The invention relates to a composition energizing and stimulating the gonads and cognitive activities, prescribed in sustaining efforts and in promoting the recovery of individuals working under phys. and psychical stress conditions. According to the invention, the composition comprises citric acid, sucrose, taurine, vitamin B6, vitamin B12, folic acid, lactic acid, caffeine, calcium fructoborate and water.

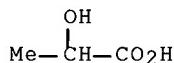
CC 63-6 (Pharmaceuticals)
 Section cross-reference(s): 1

IT **Cognition enhancers**
 Reproductive system
 (composition energizing and stimulating gonadal and cognitive activities)

IT 50-21-5, Lactic acid, biological studies 57-50-1, Sucrose, biological studies 58-08-2, Caffeine, biological studies 59-30-3, Folic acid 68-19-9, Vitamin B12 77-92-9, Citric acid, biological studies 107-35-7, Taurine 8059-24-3, Vitamin B6 250141-42-5, Calcium fructoborate
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (composition energizing and stimulating gonadal and cognitive activities)

IT 50-21-5, Lactic acid, biological studies
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (composition energizing and stimulating gonadal and cognitive activities)

RN 50-21-5 HCAPLUS
 CN Propanoic acid, 2-hydroxy- (CA INDEX NAME)



L45 ANSWER 2 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2006:708410 HCPLUS Full-text
 DOCUMENT NUMBER: 145:152735
 TITLE: Method and compositions for potentiating pharmaceuticals with amino acid-based medical foods
 INVENTOR(S): Shell, William E.; Charuvastra, Elizabeth
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 228,765,.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006159726	A1	20060720	US 2006-386325	20060322 <--
US 2004043054	A1	20040304	US 2002-228765	20020827 <--
PRIORITY APPLN. INFO.:			US 2002-228765	A2 20020827 <--

AB The methods and compns. for potentiating pharmaceuticals with amino acid-based medical foods provides improved cognitive function; induced and maintained sleep; reduced pain, inflammation, blood pressure, anxiety, asthma, duration of viral infection, insulin resistance, and appetite; and treated depression. The amino acid-based medical foods, co-packed with at least one selected pharmaceutical, potentiate the pharmaceutical by enhancing the production of neurotransmitters through the oral administration of neurotransmitter precursors, along with natural plant and animal substances that stimulate uptake of the neurotransmitter precursors, cause release of neurotransmitters, cause disinhibition of the neuronal brake, and activate adenylylate cyclase in order to avoid tachyphylaxis and prevent pharmacol. tolerance.

INCL 424439000; 424728000; 424730000; 424739000; 424752000; 424769000; 514400000; 514263310; 514561000; 514649000

CC 63-6 (Pharmaceuticals)
 Section cross-reference(s): 1, 2, 18

IT Analgesics
 Anti-inflammatory agents
 Antiasthmatics
 Antidepressants
 Antihypertensives
 Anxiety
 Anxiolytics
 Appetite
 Asthma
 Blood pressure
 Cinnamon (spice)
 Cocoa products
Cognition enhancers
Cognitive disorders
 Diabetes mellitus
 Drug tolerance
 Eating disorders
 Food

Ginkgo biloba
 Heart rate
 Human
 Hypericum perforatum
 Hypertension
 Hypnotics and Sedatives
 Memory disorders
 Obesity
 Pain
 Parasympathetic nervous system
 Sleep
 Sleep disorders
 Sympathetic nervous system
 Tachyphylaxis
 (method and compns. for potentiating pharmaceuticals with amino acid-based medical foods)
 IT **56-84-8**, Aspartic acid, biological studies **56-85-9**,
 Glutamine, biological studies 58-08-2, Caffeine, biological studies
28805-76-7, Aminobutyric acid
 RL: **FFD (Food or feed use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (method and compns. for potentiating pharmaceuticals with amino acid-based medical foods)
 IT 50-55-5, Reserpine 50-78-2, Aspirin 51-63-8, Dexedrine 52-01-7,
 Spironolactone 54-31-9, Furosemide 55-63-0, Nitroglycerin
56-86-0, L-Glutamic acid, biological studies 57-27-2, Morphine,
 biological studies 57-33-0 57-42-1, Meperidine 57-53-4, Meprobamate
 58-25-3, Chlordiazepoxide 58-55-9, Theophylline, biological studies
 58-93-5, Hydrochlorothiazide 60-18-4, Tyrosine, biological studies
 62-49-7, Choline 71-00-1, Histidine, biological studies 72-44-6,
 Methaqualone 73-22-3, Tryptophan, biological studies 74-79-3,
 Arginine, biological studies 76-57-3, Codeine 76-73-3, Secobarbital
 77-21-4, Glutethimide 77-36-1, Chlorthalidone 90-82-4, Pseudoephedrine
 94-07-5, Synephrine 113-18-8, Ethchlorvynol 113-45-1, Methylphenidate
 122-09-8, Phentermine 125-29-1, Hydrocodone 299-42-3, Ephedrine
 300-62-9D, Amphetamine, derivs. 302-17-0, Chloral hydrate 304-20-1,
 Hydralazine hydrochloride 318-98-9, Propranolol hydrochloride
 396-01-0, Triamterene 438-41-5, Chlordiazepoxide hydrochloride
 439-14-5, Diazepam 466-99-9, Hydromorphone 469-62-5, Propoxyphene
 555-30-6, Alpha methyldopa 657-24-9, Metformin 768-94-5, Amantadine
 846-50-4, Temazepam 1115-70-4, Glucophage 2016-88-8, Amiloride
 hydrochloride 4350-09-8, 5-Hydroxytryptophan 7491-74-9, Piracetam
 17560-51-9, Metolazone 19237-84-4, Prazosin hydrochloride 19794-93-5,
 Trazodone 21829-25-4, Nifedipine 22204-53-1, Naproxen 27203-92-5,
 Tramadol 28981-97-7, Alprazolam 29122-68-7, Atenolol 29975-16-4,
 Estazolam 32780-64-6, Labetalol hydrochloride 33286-22-5, Diltiazem
 hydrochloride 37517-30-9, Acebutolol 42200-33-9, Nadolol 51781-21-6,
 Carteolol hydrochloride 54739-18-3, Fluvoxamine 54910-89-3, Fluoxetine
 55985-32-5, Nicardipine 56392-17-7, Metoprolol tartrate 57109-90-7,
 Chlorazepate 59729-33-8, Citalopram 60142-96-3, Gabapentin
 61869-08-7, Paroxetine 62571-86-2, Captopril 63074-08-8, Terazosin
 hydrochloride 63659-18-7, Betaxolol 68693-11-8, Provigil 72509-76-3,
 Felodipine 72956-09-3, Carvedilol 75695-93-1, Isradipine 76095-16-4,
 Enalapril maleate 76547-98-3, Lisinopril 77883-43-3, Doxazosin
 mesylate 79617-96-2, Sertraline 82586-55-8, Quinapril hydrochloride
 86541-74-4, Benazepril hydrochloride 87333-19-5, Ramipril 87679-37-6,
 Trandolapril 88889-14-9, Fosinopril sodium 98418-47-4, Metoprolol
 succinate 99294-93-6, Zolpidem tartrate 103775-10-6, Moexipril
 104344-23-2, Bisoprolol fumarate 111470-99-6, Amlodipine besylate

116539-59-4, Duloxetine 124750-99-8, Losartan potassium 124832-27-5,
 Valtrex 128196-01-0, Escitalopram 137862-53-4, Valsartan
 138402-11-6, Irbesartan 138729-47-2, Eszopiclone 139481-59-7,
 Candesartan 151319-34-5, Zaleplon 162011-90-7, Rofecoxib
 169590-42-5, Celecoxib 171599-83-0, Viagra
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (method and compns. for potentiating pharmaceuticals with amino acid-based medical foods)

IT 56-84-8, Aspartic acid, biological studies 56-85-9,
 Glutamine, biological studies 28805-76-7, Aminobutyric acid
 RL: **FFD (Food or feed use); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**

(method and compns. for potentiating pharmaceuticals with amino acid-based medical foods)

RN 56-84-8 HCPLUS

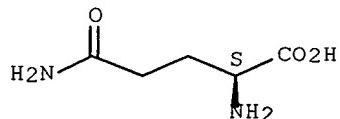
CN L-Aspartic acid (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).

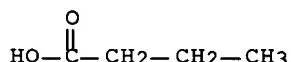


RN 56-85-9 HCPLUS
 CN L-Glutamine (CA INDEX NAME)

Absolute stereochemistry.



RN 28805-76-7 HCPLUS
 CN Butanoic acid, amino- (9CI) (CA INDEX NAME)

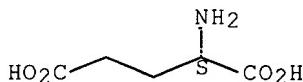


D1-NH2

IT 56-86-0, L-Glutamic acid, biological studies
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (method and compns. for potentiating pharmaceuticals with amino acid-based medical foods)

RN 56-86-0 HCAPLUS
 CN L-Glutamic acid (CA INDEX NAME)

Absolute stereochemistry.



L45 ANSWER 3 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2006:465304 HCAPLUS Full-text
 DOCUMENT NUMBER: 144:481065
 TITLE: Carbonic anhydrase activators for enhancing learning and memory
 INVENTOR(S): Alkon, Daniel; Sun, Miao-Kun
 PATENT ASSIGNEE(S): Blanchette Rockefeller Neurosciences Institute, USA
 SOURCE: PCT Int. Appl., 33 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002087423	A2	20021107	WO 2002-US13784	20020502 <--
WO 2002087423	A3	20030227		
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CA 2446074	A1	20021107	CA 2002-2446074	20020502 <--
EP 1383497	A2	20040128	EP 2002-725885	20020502 <--
			R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR	
JP 2004535386	T	20041125	JP 2002-584781	20020502 <--
US 2004235889	A1	20041125	US 2004-476459	20040622 <--
PRIORITY APPLN. INFO.:			US 2001-287721P	P 20010502 <--
			WO 2002-US13784	W 20020502 <--

OTHER SOURCE(S): MARPAT 144:481065

AB The invention provides methods for improving attention and/or memory acquisition comprising stimulating intraneuronal carbonic anhydrase activity. The stimulation is achieved by administering a carbonic anhydrase activator. The method allows treating neurodegenerative disorders to enhance cognitive ability, treating dementia, and also enhancing attention and learning in healthy individuals. The invention provides a method for improving attentive cognition comprising administering a compound that potentiates intraneuronal carbonic anhydrase activity thereby improving establishment of a theta rhythm.

IC ICM A61B

CC 1-11 (Pharmacology)

Section cross-reference(s): 63

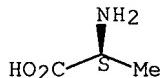
IT Alzheimer's disease
Anti-Alzheimer's agents
Cognition
Cognition enhancers
Drug delivery systems
Learning
Memory, biological
(carbonic anhydrase activators for enhancing learning and memory by establishment of theta rhythm in relation to treating neurodegenerative disorders)

IT 51-45-6, 1H-Imidazole-4-ethanamine, biological studies 51-61-6, biological studies 51-65-0 **56-41-7**, L-Alanine, biological studies 60-18-4, L-Tyrosine, biological studies 63-84-3 **63-91-2**, L-Phenylalanine, biological studies 64-04-0, Benzeneethanamine 71-00-1, L-Histidine, biological studies 75-04-7D, Ethylamine, derivs. 138-65-8 288-32-4, Imidazole, biological studies 288-32-4D, Imidazole, derivs. 329-65-7 616-47-7 2922-41-0 3387-86-8 5709-61-5 6539-57-7 7098-07-9 36507-31-0 37306-44-8, Triazole 53214-57-6 70780-90-4 82410-58-0 84661-56-3 111128-04-2
RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
(carbonic anhydrase activators for enhancing learning and memory by establishment of theta rhythm in relation to treating neurodegenerative disorders)

IT **56-41-7**, L-Alanine, biological studies **63-91-2**, L-Phenylalanine, biological studies
RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
(carbonic anhydrase activators for enhancing learning and memory by establishment of theta rhythm in relation to treating neurodegenerative disorders)

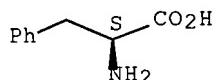
RN 56-41-7 HCPLUS
CN L-Alanine (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



RN 63-91-2 HCPLUS
CN L-Phenylalanine (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



DOCUMENT NUMBER: 144:135267
 TITLE: Mental agility lozenge, edible strip, food or drink
 INVENTOR(S): McCleary, Edward Larry
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 19 pp., Cont.-in-part of U.S.
 Ser. No. 49,244.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 12
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006014773	A1	20060119	US 2005-223719	20050909 <--
US 2002182196	A1	20021205	US 2001-837562	20010419 <--
US 6964969	B2	20051115		
US 2004043013	A1	20040304	US 2003-462958	20030617 <--
US 2005025812	A1	20050203	US 2003-616674	20030710
US 2005002992	A1	20050106	US 2004-890067	20040712
US 2006110477	A1	20060525	US 2005-49237	20050202
US 2006110478	A1	20060525	US 2005-49244	20050202
WO 2006057893	A2	20060601	WO 2005-US41765	20051117
WO 2006057893	A3	20060824		
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PRIORITY APPLN. INFO.:			US 2001-837562	A2 20010419 <--
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			US 2004-536286P	P 20040113
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			US 2004-630529P	P 20041122
			US 2005-49244	A2 20050202
			US 2000-749584	A2 20001228 <--
			US 2005-49237	A 20050202

AB A mental agility composition is composed of at least one agent which promotes synthesis of ATP and/or creatine phosphate in the body; at least one antioxidant for scavenging free radicals in at least one pathway in the body; at least one agent for normalizing or maintaining membrane function and structure in the body; at least one agent for normalizing or maintaining normal neurotransmitter function in the body; at least one agent for down-regulating cortisol action; and at least one agent for suppressing activation of apoptotic pathways in the body. The composition may further contain one or more of at least one agent for suppressing inflammation in the body; at least one agent for normalizing or maintaining vascular wall function and structure in the body; at least one agent for normalizing or maintaining function of nerve growth factors and/or neurotropic factors in the body; at least one agent for suppressing toxic metal ionic effects; at least one agent for normalizing or maintaining Me metabolism in the body; at least one agent for

normalizing or maintaining metabolism of insulin and glucose in the body; and at least one agent for up-regulating activity of heat shock proteins in the body. The composition is administered in the form of a breath-care strip, mint or lozenge, or a food or beverage product.

INCL 514283000; 514440000; 514350000; 514553000; 424094100; 424752000;
514561000; 514565000; 514733000; 514763000

CC 63-6 (Pharmaceuticals)

IT Beverages

Candy

Cognition enhancers

Drug delivery systems

Food

Ginkgo biloba

Human

Radical scavengers

(mental agility lozenge, edible strip, food or drink)

IT 62-49-7, Choline 65-23-6, Pyridoxine 87-89-8, Inositol 107-35-7,
Taurine 107-43-7, Trimethylglycine 303-98-0, Coenzyme q10
501-36-0, Resveratrol 502-65-8, Lycopene 1200-22-2, α Lipoic
acid 3040-38-8, Acetyl L carnitine 6020-87-7, Creatine monohydrate
42971-09-5, Vinpocetine 102518-79-6, Huperzine A
RL: **THU (Therapeutic use); BIOL (Biological study);**

USES (Uses)

(mental agility lozenge, edible strip, food or drink)

IT 107-43-7, Trimethylglycine

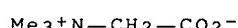
RL: **THU (Therapeutic use); BIOL (Biological study);**

USES (Uses)

(mental agility lozenge, edible strip, food or drink)

RN 107-43-7 HCPLUS

CN Methanaminium, 1-carboxy-N,N,N-trimethyl-, inner salt (CA INDEX NAME)



L45 ANSWER 5 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2006:31909 HCPLUS Full-text
 DOCUMENT NUMBER: 144:121848
 TITLE: Hydrazino compounds. for controlling damage mediated
by alpha, beta-unsatd. aldehydes
 INVENTOR(S): Burcham, Philip C.; Pyke, Simon M.; Kaminskas, Lisa
M.; Musgrave, Ian
 PATENT ASSIGNEE(S): Adelaide Research & Innovation Pty. Ltd., Australia
 SOURCE: PCT Int. Appl., 132 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 3
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006002473	A1	20060112	WO 2005-AU967	20050701
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA,				

NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,
 SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU,
 ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
 IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
 CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
 GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
 KG, KZ, MD, RU, TJ, TM

US 2006160848	A1	20060720	US 2004-882187	20040702 <--
PRIORITY APPLN. INFO.:			US 2004-882187	A 20040702
			AU 2002-9813	A 20020104 <--
			AU 2002-1415	A 20020328 <--
			WO 2002-AU900	A2 20020705 <--

OTHER SOURCE(S): MARPAT 144:121848

AB This invention relates to a method of preventing and/or treating a disease or condition associated with damage mediated by an -unsatd. aldehyde in a subject, the method including the step of administering to the subject a therapeutically effective amount of a hydrazino compound. The cytoprotective activity of hydrazino compounds. was demonstrated in in vitro and in vivo studies of α,β -unsatd. aldehyde-induced hepatotoxicity. Acrolein-adducted protein trapping by hydralazine in mouse liver was accompanied by increased hepatoprotection.

IC ICM A61K031-15
 ICS A61P025-28

CC 1-12 (Pharmacology)

Section cross-reference(s): 4

IT Aging, animal

Alzheimer's disease

Anti-Alzheimer's agents

Antioxidants

Antiparkinsonian agents

Bioassay

Central nervous system agents

Cognition enhancers

Cognitive disorders

Diagnosis

Hepatotoxicity

Neurotoxicity

Nucleophilicity

Oxidative stress, biological

Parkinson's disease

(hydrazino compounds. for controlling damage mediated by alpha,
 beta-unsatd. aldehydes)

IT 56-87-1, Lysine, biological studies

RL: **BSU (Biological study, unclassified); BIOL (Biological study)**

(significance in acrolein-induced RNase crosslinking; hydrazino compounds. for controlling damage mediated by alpha, beta-unsatd. aldehydes)

IT 56-87-1, Lysine, biological studies

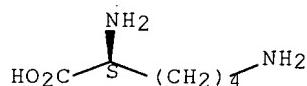
RL: **BSU (Biological study, unclassified); BIOL (Biological study)**

(significance in acrolein-induced RNase crosslinking; hydrazino compounds. for controlling damage mediated by alpha, beta-unsatd. aldehydes)

RN 56-87-1 HCPLUS

CN L-Lysine (CA INDEX NAME)

Absolute stereochemistry.



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 6 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2005:1310905 HCPLUS Full-text
 DOCUMENT NUMBER: 144:45513
 TITLE: Composition comprising Xanthoceras sorbifolia extracts, compounds isolated from same, methods for preparing same, and uses thereof
 INVENTOR(S): Chan, Pui-Kwong; Mak, May Sung; Wang, Yun
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 194 pp., Cont.-in-part of U.S. Ser. No. 906,303.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 11
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005276872	A1	20051215	US 2005-117760	20050427 <--
US 2003091669	A1	20030515	US 2001-944805	20010831 <--
US 6616943	B2	20030909		
WO 2003017919	A2	20030306	WO 2002-IB4750	20020828 <--
WO 2003017919	A3	20040722		
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US 2004146591	A1	20040729	US 2003-471384	20030904 <--
US 7189420	B2	20070313		
WO 2005037200	A2	20050428	WO 2004-US33359	20041008
WO 2005037200	A3	20050616		
WO 2005037200	A8	20050901		
WO 2005037200	B1	20051006		
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WO 2005063273	A1	20050714	WO 2004-US43465	20041223
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US 2005220910	A1	20051006	US 2005-906303	20050214
WO 2006029221	A2	20060316	WO 2005-US31900	20050907
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US 2006111310	A1	20060525	US 2005-267523	20051104
US 2006122129	A1	20060608	US 2005-289142	20051128
WO 2006116656	A2	20061102	WO 2006-US16158	20060427
WO 2006116656	A3	20070215		
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US 2006263458	A1	20061123	US 2006-412659	20060427
PRIORITY APPLN. INFO.:			US 2001-944805	A2 20010831 <--
			WO 2002-IB4750	W 20020828 <--
			US 2003-471384	A2 20030904
			US 2003-509851P	P 20031009
			US 2003-532101P	P 20031223
			US 2004-607858P	P 20040907
			US 2004-613811P	P 20040927
			US 2004-617379P	P 20041008
			WO 2004-US33359	A2 20041008
			WO 2004-US43465	A2 20041223
			US 2005-906303	A2 20050214
			US 2005-117760	A 20050427
			US 2005-675282P	P 20050427
			US 2005-675284P	P 20050427
			US 2005-131551	A 20050517
			WO 2005-US31900	A2 20050907
			US 2005-267523	A2 20051104

OTHER SOURCE(S): MARPAT 144:45513

AB This invention provides compns., methods and process of producing exts. and pure compds. from Xanthoceras sorbifolia. The extract comprises saponins and other constituents including alkaloids, coumarins, saccharides, proteins, polysaccharides, glycosides, tannins, acid, flavonoids and others. The composition can be used for treating cancer and other conditions, such as arthritis, rheumatism, poor circulation, arteriosclerosis, Raynaud's syndrome, angina pectoris, cardiac disorder, coronary heart disease, headache, kidney disorder, and impotence; for improving cerebral functions; or for curing enuresis, frequent micturition, urinary incontinence, dementia, weak intelligence and Alzheimer's disease, autism, brain trauma, Parkinson's, cerebral dysfunctions, and treating arthritis, rheumatism, poor circulation, arteriosclerosis, Raynaud's syndrome, angina pectoris, cardiac disorder, headache, dizziness, kidney disorder. This invention provides compds. of oleanene triterpenoidal saponin in nature with the characteristics that at least one angeloyl group attache to Carbon 21 or/and 22, or/and linked to the sugar. The compds. of the present invention have various pharmaceutical and therapeutic applications.

IC ICM A61K035-78

INCL 424767000; 424769000

CC 1-12 (Pharmacology)

Section cross-reference(s): 11, 63

IT AIDS (disease)

Aging, animal

Alzheimer's disease

Amnesia

Angelica sinensis

Anti-AIDS agents

Anti-Alzheimer's agents

Anti-inflammatory agents

Antiarteriosclerotics

Antiarthritics

Anticholesteremic agents

Antioxidants

Antiparkinsonian agents

Antirheumatic agents

Antitumor agents

Antiviral agents

Armillaria mellea

Arteriosclerosis

Arthritis

Bladder, neoplasm

Bone, neoplasm

Brain, neoplasm

Cardiovascular agents

Clausena lansium

Cognition enhancers

Combination chemotherapy

Cordyceps

Dietary fiber

Dizziness

Drug delivery systems

Echinacea

Extraction

Ganoderma

Gastrodia elata

Ginkgo

Glycyrrhiza uralensis

HPLC

Headache
 Health food
 Heart, disease
Hedysarum polybotrys
 Human
 Human immunodeficiency virus
 Hypercholesterolemia
Hypericum perforatum
 Hypertriglyceridemia
 Hypolipemic agents
 Inflammation
 Kidney, disease
 Learning
 Leukemia
 Liver, neoplasm
 Lung, neoplasm
 Mammary gland, neoplasm
 Memory disorders
 Micturition
Monascus
 Neoplasm
 Nervous system agents
 Ovary, neoplasm
Panax ginseng
Panax notoginseng
Panax quinquefolium
 Parkinson's disease
Polygala tenuifolia
Poria cocos
 Prostate gland, neoplasm
Pueraria lobata
Rehmannia glutinosa
 Rheumatic diseases
Salvia miltiorrhiza
 Signal transduction, biological
 Skin, neoplasm
 Stress, biological
Xanthoceras sorbifolia

(*Xanthoceras sorbifolia* extract composition, isolated compds., preparation methods,
 and therapeutic use)

IT 60-29-7, Ether, biological studies 64-17-5, Ethanol, biological studies
 67-56-1, Methanol, biological studies 67-64-1, Acetone, biological
 studies 67-66-3, Chloroform, biological studies 71-36-3, n-Butanol,
 biological studies 71-43-2, Benzol, biological studies 108-88-3,
 Toluol, biological studies 110-54-3, n-Hexane, biological studies
141-78-6, Ethyl acetate, biological studies 7732-18-5, Water,
 biological studies

RL: **BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)**

(extraction with; *Xanthoceras sorbifolia* extract composition, isolated
 compds., preparation methods, and therapeutic use)

IT **141-78-6**, Ethyl acetate, biological studies
 RL: **BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)**

(extraction with; *Xanthoceras sorbifolia* extract composition, isolated
 compds., preparation methods, and therapeutic use)

RN 141-78-6 HCAPLUS

CN Acetic acid ethyl ester (CA INDEX NAME)

Et—O—Ac

L45 ANSWER 7 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2005:572580 HCAPLUS Full-text
 DOCUMENT NUMBER: 143:53546
 TITLE: Compositions and methods to treat recurrent medical conditions
 INVENTOR(S): Patrick, Jason; Davis, Michael
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 18 pp.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 5
 PATENT INFORMATION:

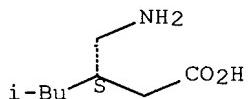
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005143314	A1	20050630	US 2004-24921	20041229
AU 2004311869	A1	20050721	AU 2004-311869	20041229
WO 2005065308	A2	20050721	WO 2004-US43628	20041229
WO 2005065308	A3	20051208		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1703907	A2	20060927	EP 2004-815650	20041229
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US 2006252761	A1	20061109	US 2006-347937	20060206 <--
PRIORITY APPLN. INFO.:			US 2003-533003P	P 20031229
			US 2004-625253P	P 20041105
			US 2001-279868P	P 20010329 <--
			US 2002-363991P	P 20020313 <--
			WO 2002-US9467	W 20020328 <--
			US 2003-492795P	P 20030806
			US 2004-473640	A2 20040422
			WO 2004-US24841	A2 20040803
			US 2004-924591	A2 20040824
			US 2004-24921	A2 20041229
			WO 2004-US43628	W 20041229
			US 2005-651114P	P 20050208
			US 2005-667140P	P 20050331

AB The invention describes methods and compns. for alleviating recurrent medical afflictions for which anxiety may cause or exacerbate the affliction. A subject suffering from the affliction is treated with a combination of a

pharmaceutical that enhances learning, and a second pharmaceutical recognized to be useful for treatment of the affliction. Representative afflictions include insomnia, erectile dysfunction, female sexual dysfunction, neuropathic pain, attention deficit disorder, and depression.

- IC ICM A61K038-09
 ICS A61K031-56; A61K031-57; A61K031-519; A61K031-496; A61K031-473;
 A61K031-137
- INCL 514017000; 514170000; 514252160; 514295000; 514649000; 514290000;
 514469000; 514301000
- CC 1-11 (Pharmacology)
 Section cross-reference(s): 2, 18
- IT Antidepressants
 Anxiety
 Anxiolytics
Cognition enhancers
 Human
 Insomnia
 Learning disorders
 (compns. and methods to treat recurrent medical conditions for which anxiety may cause or exacerbate such condition)
- IT 50-28-2, Estradiol, biological studies 50-47-5, Desipramine 50-48-6,
 Amitriptyline 51-64-9, Dextroamphetamine 58-00-4, Apomorphine
 58-22-0, Testosterone 68-41-7, D-Cycloserine 72-69-5, Nortriptyline
 113-45-1, Methylphenidate 300-62-9, Amphetamine 549-18-8,
 Amitriptyline hydrochloride 604-75-1, Oxazepam 846-49-1, Lorazepam
 846-50-4, Temazepam 1622-61-3, Clonazepam 17617-23-1, Flurazepam
 28911-01-5, Triazolam 28981-97-7, Alprazolam 34911-55-2, Bupropion
 43200-80-2, Zopiclone 54739-18-3, Fluvoxamine 54910-89-3, Fluoxetine
 59729-33-8, Citalopram 60142-96-3, Gabapentin 61869-08-7, Paroxetine
 79617-96-2, Sertraline 82626-48-0, Zolpidem 83015-26-3, Atomoxetine
 138729-47-2, Eszopiclone 139755-83-2, Sildenafil **148553-50-8**,
 Pregabalin 151319-34-5, Zaleplon 171596-29-5, Tadalafil 189691-06-3,
 PT-141 224785-90-4, Vardenafil 325715-02-4, Indiplon
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (compns. and methods to treat recurrent medical conditions for which anxiety may cause or exacerbate such condition)
- IT **148553-50-8**, Pregabalin
 RL: **PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)**
 (compns. and methods to treat recurrent medical conditions for which anxiety may cause or exacerbate such condition)
- RN 148553-50-8 HCPLUS
 CN Hexanoic acid, 3-(aminomethyl)-5-methyl-, (3S)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L45 ANSWER 8 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2005:546883 HCPLUS Full-text
 DOCUMENT NUMBER: 143:65362
 TITLE: Therapeutic placebo enhancement of commonly used

DOCUMENT TYPE:

Journal

LANGUAGE:

Russian

AB The membranotropic action of antiarrhythmic and nootropic drugs (anaprilin, etmozin, carnitine, piracetam, etc.) was studied on liposomes from lecithin, cardiolipin, and rabbit brain synaptosomal membranes by spectrophotometry and spectrofluorometry using pyrene fluorescent probes. The ability of these compds. to form complexes with lipid bilayer, increasing its motility, was shown. The studied physiol. active compds. decrease calcium-binding properties of synaptosomal membranes and of the cardiolipin bilayer.

CC 1-4 (Pharmacology)

IT Antiarrhythmics

Liposome

Membrane, biological

Nootropics

(influence of antiarrhythmic and nootropic drugs on motility and calcium-binding properties of model and biol. membranes)

IT 69-89-6, Xanthine 253-82-7, Quinazoline 288-88-0, 1,2,4-Triazole 318-98-9, Anaprilin 541-15-1, Carnitine 7491-74-9, Piracetam 29560-58-5, Etmozin 158701-37-2, Fenquizol

RL: **BAC** (Biological activity or effector, except adverse);
BSU (Biological study, unclassified); **Biol** (Biological study)

(influence of antiarrhythmic and nootropic drugs on motility and calcium-binding properties of model and biol. membranes)

IT 541-15-1, Carnitine

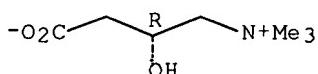
RL: **BAC** (Biological activity or effector, except adverse);
BSU (Biological study, unclassified); **Biol** (Biological study)

(influence of antiarrhythmic and nootropic drugs on motility and calcium-binding properties of model and biol. membranes)

RN 541-15-1 HCAPLUS

CN 1-Propanaminium, 3-carboxy-2-hydroxy-N,N,N-trimethyl-, inner salt, (2R)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L45 ANSWER 215 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1994:280260 HCAPLUS Full-text

DOCUMENT NUMBER: 120:280260

TITLE: Agent with adaptogenic activity

PATENT ASSIGNEE(S): "Sojuzpotent", Moscow, Russia

SOURCE: PCT Int. Appl.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Russian

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9400116	A1	19940106	WO 1993-RU138	19930628 <-- W: AT, AU, BB, BG, BR, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP,

KR, LK, LU, MG, MN, MW, NL, NO
 RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
 RU 2038077 C1 19950627 RU 1992-5049402 19920626 <--
 AU 9345919 A 19940124 AU 1993-45919 19930628 <--
 EP 605742 A1 19940713 EP 1993-916319 19930628 <--
 R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, NL, SE
 JP 07500846 T 19950126 JP 1993-502231 19930628 <--
 HU 71836 A2 19960228 HU 1994-574 19930628 <--
 NO 9400692 A 19940405 NO 1994-692 19940228 <--
 PRIORITY APPLN. INFO.: SU 1992-5049402 A 19920626 <--
 RU 1992-5049402 A 19920626 <--
 RU 20 5-5049402 A 19920626 <--
 WO 1993-RU138 A 19930628 <--

AB The invention relates to medicine, in particular, to exptl. and clin. therapy and may be used for adaptogenic treatment of humans subjected to extreme conditions or recovering from a serious illness. The synthetic amino acid DL-valine can be used as an agent with adaptogenic activity. DL-valine, causing a state of nonspecific heightened resistance of the body, especially the human body, increases mental and phys. working capacity as well as normalizes psychol. activity. Its influence is also characterized by a significant increase of the immune protection of the organism, disappearance of some chronic inflammations, normalization of the metabolism and of the functional state of the liver, and of the cardiovascular system. The proposed agent is easily synthetically obtained and functions as a nutritional component of the human food ration with no adverse side effect and may be widely used for prophylaxis as well as phys. and psychol. care. Thus, a postmenopausal 55-yr-old woman presenting with hypertension, stenocardia, depression, complications of influenza, and who was overweight and fatigued quickly during mental or phys. work, was given 0.5 g DL-valine each evening in a half-glass of warm milk for 2 mo. After 2 wk, blood pressure normalized and remained so during the period of study. The symptoms of stenocardia disappeared completely after 1 mo and have not returned. Psychol. she improved greatly and the influenza complications disappeared. Her capacity for phys. and mental work increased markedly, and her weight began to normalize without special dieting.

IC A61K031-195

CC 63-2 (Pharmaceuticals)

IT Antidepressants

Antihypertensives

Antiobesity agents

Nootropics

(racemic valine as, in humans)

IT 516-06-3, DL-Valine

RL: **BAC (Biological activity or effector, except adverse);**

BSU (Biological study, unclassified); BIOL (Biological study)

(adaptogenic activity of, in humans with mental and phys. disorders)

IT 516-06-3, DL-Valine

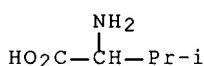
RL: **BAC (Biological activity or effector, except adverse);**

BSU (Biological study, unclassified); BIOL (Biological study)

(adaptogenic activity of, in humans with mental and phys. disorders)

RN 516-06-3 HCPLUS

CN Valine (CA INDEX NAME)



L45 ANSWER 216 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1994:25428 HCAPLUS Full-text
 DOCUMENT NUMBER: 120:25428
 TITLE: Effects of the nootropic AWD 52-39 on the blood-brain transfer of leucine, choline and glucose in rats after 14-d exposure to ethanol
 AUTHOR(S): Brust, P.; Jordan, Kirsten
 CORPORATE SOURCE: Dep. Cell Biol. Regul., Univ. Leipzig, Germany
 SOURCE: Pharmazie (1992), 47(8), 616-20
 CODEN: PHARAT; ISSN: 0031-7144
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The transport of the neutral amino acid L-leucine as well as of choline and D-glucose across the blood-brain barrier (BB) of male Wistar rats was studied after 14-day exposure to ethanol and treatment with the nootropic drug AWD 52-39(1). After ethanol exposure the half-saturation constant (Km) and the maximum velocity of transport (Vmax) declined in the majority of the investigated brain regions. Also, the treatment elicited a regionally different increase of the permeability-surface area (PS) product of choline (between 10% and 33%) and glucose (between 12% and 27%). The changes in the blood-brain transfer of the three compds. were diminished or prevented by addnl. application of l. The cerebral blood flow was increased by the exposure to ethanol maximally by 44%. After addnl. administration of l the changes were reversed and the blood flow reached control values. In addition, the activity of the enzyme acetylcholine esterase was determined in the striatum and the hippocampus. After ethanol exposure the enzyme activity declined by 32%. It was less diminished after treatment with l. The latter effects let one assume that the changes in the BBB permeability elicited by ethanol and l are related to alterations of brain metabolism

CC 4-7 (Toxicology)
 Section cross-reference(s): 1

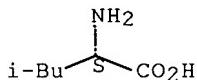
IT **Nootropics**
 (AWD 52-39 as, prevention of ethanol-induced brain injury)

IT 50-99-7, Glucose, biological studies **61-90-5**, Leucine,
 biological studies 62-49-7, Choline
 RL: **BIOL (Biological study)**
 (AWD 52-39 effect on ethanol-induced blood-brain barrier permeability to)

IT **61-90-5**, Leucine, biological studies
 RL: **BIOL (Biological study)**
 (AWD 52-39 effect on ethanol-induced blood-brain barrier permeability to)

RN 61-90-5 HCPLUS
 CN L-Leucine (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L45 ANSWER 217 OF 220 HCAPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1993:641401 HCAPLUS Full-text
 DOCUMENT NUMBER: 119:241401

TITLE: Pharmaceutical preparation of antistress,
stress-preventive and nootropic (psychotropic) action
INVENTOR(S): Komissarova, I. A.; Gudkova, Yu. V.; Soldatenkova, T.
D.; Kondrashova, T. T.; Burbenskaya, N. M.
PATENT ASSIGNEE(S): Russia
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Russian
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9313764	A1	19930722	WO 1992-RU5	19920110 <--
W: AU, BR, CA, HU, JP, KR, RU, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG				
AU 9211928	A	19930803	AU 1992-11928	19920110 <--
EP 575613	A1	19931229	EP 1992-903883	19920110 <--
EP 575613	B1	19980722		
R: AT, CH, DE, FR, GB, LI, SE				
JP 06505014	T	19940609	JP 1992-504720	19920110 <--
JP 3162709	B2	20010508		
AT 168559	T	19980815	AT 1992-903883	19920110 <--
CN 1050993	B	20000405	CN 1993-100251	19930109 <--
US 5643954	A	19970701	US 1993-119050	19930910 <--
US 5731349	A	19980324	US 1996-701147	19960821 <--
PRIORITY APPLN. INFO.:			EP 1992-903883	A 19920110 <--
			WO 1992-RU5	A 19920110 <--
			US 1993-119050	A3 19930910 <--
			US 1996-701147	A 19960821 <--

AB A pharmaceutical preparation containing glycine or its salt is useful for stress prevention and has nootropic activity and can be administered sublingually. The effectiveness to the compound in improving the mental working capacity in students was demonstrated. The indexed measured in the stress test were blood pressure and pulse values.

IC ICM A61K031-195

ICS A61K009-20; A61K047-38

CC 1-11 (Pharmacology)

Section cross-reference(s): 63

IT **Nootropics**

(glycine pharmaceuticals, stress prevention by)

IT 56-40-6, Glycine, biological studies

RL: **Biol (Biological study)**

(pharmaceuticals, nootropic activity of and stress prevention by)

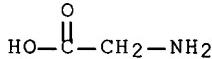
IT 56-40-6, Glycine, biological studies

RL: **Biol (Biological study)**

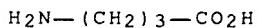
(pharmaceuticals, nootropic activity of and stress prevention by)

RN 56-40-6 HCPLUS

CN Glycine (CA INDEX NAME)



L45 ANSWER 218 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1993:574100 HCPLUS Full-text
 DOCUMENT NUMBER: 119:174100
 TITLE: DM-9384, a new cognition-enhancing agent, increases the turnover of components of the GABAergic system in the rat cerebral cortex
 AUTHOR(S): Watabe, Shigeo; Yamaguchi, Hitoshi; Ashida, Shinichiro
 CORPORATE SOURCE: Explorat. Res. Lab. II, Daiichi Pharm. Co., Ltd., Tokyo, 134, Japan
 SOURCE: European Journal of Pharmacology (1993), 238(2-3), 303-9
 CODEN: EJPRAZ; ISSN: 0014-2999
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB DM-9384 (nefiracetam) (N-(2,6-dimethylphenyl)-2-(2-oxo-1-pyrrolidinyl)acetamide), a pyrrolidone derivative (or a cyclic derivative of γ -aminobutyric acid (GABA)), is a newly developed nootropic (or cognition-enhancing) agent. In the present study, the authors examined the biochemical effect of DM-9384 on GABAergic neurons in adult rat brains. DM-9384, when administered orally at a daily dose of 10 mg/kg for 7 days, significantly increased GABA turnover and glutamic acid decarboxylase activity in the cortex and hippocampus, and stimulated Na⁺-dependent high-affinity GABA uptake in cortical synaptosomes. In *in vitro* experiments, the K⁺-evoked release of [14C]GABA from cortical slices was markedly increased by low concns. (10⁻⁸, 10⁻⁹ M) of DM-9384. The binding of GABA and benzodiazepine to their receptors in the brain was not affected by DM-9384 (10⁻¹⁰-10⁻³ M). The results suggest that DM-9384 increases the turnover of components of the GABAergic system by influencing presynaptic sites rather than postsynaptic sites.
 CC 1-11 (Pharmacology)
 IT **Nootropics**
 (DM9384 as, cerebral cortex GABAergic system component turnover increase by)
 IT **Mental activity**
 (cognition, enhancer, DM9384 as, cerebral cortex GABAergic system component turnover increase by)
 IT **56-12-2, GABA, biological studies**
 RL: **Biol (Biological study)**
 (cognition enhancer DM9384 increase of turnover of, in cerebral cortex)
 IT **56-12-2, GABA, biological studies**
 RL: **Biol (Biological study)**
 (cognition enhancer DM9384 increase of turnover of, in cerebral cortex)
 RN 56-12-2 HCPLUS
 CN Butanoic acid, 4-amino- (CA INDEX NAME)



L45 ANSWER 219 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 1993:462866 HCPLUS Full-text
 DOCUMENT NUMBER: 119:62866
 TITLE: Effects of mood-stabilizing drugs on the peripheral serotonin transporter
 AUTHOR(S): Marazziti, D.; Rotondo, A.; Lenzi, A.; Presta, S.; Silvestri, S.; Cassano, G. B.
 CORPORATE SOURCE: Inst. Psychiatry, Univ. Pisa, Pisa, Italy
 SOURCE: Human Psychopharmacology (1992), 7(6),

397-401

CODEN: HUPSEC; ISSN: 0885-6222

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB The authors studied the effect of lithium (L), carbamazepine (CBZ), valproic acid (VA), verapamil (VP) and nifedipine (NF) on the specific binding of ³H-imipramine (³H-IMI) to platelet membranes, as compared with clomipramine (CLO). The results showed that VP, NF and CLO exerted a concentration-dependent inhibition on the IMI binding; CLO had the most potent inhibitory effect. No effect was observed with L, CBZ or VA. These results suggest that while VP and NF interact with the 5-HT transporter complex, L, CBZ and VA do not. Therefore, their effective (or suggested) mood-stabilizing effects are likely to be related to different mechanisms.

CC 1-11 (Pharmacology)

IT **Nootropics**

(peripheral serotonin transporter response to)

IT 52-53-9, Verapamil **99-66-1**, Valproic acid 298-46-4,
Carbamazepine 7439-93-2, Lithium, biological studies 21829-25-4,
NifedipineRL: **Biol (Biological study)**

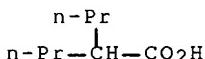
(peripheral serotonin transporter response to)

IT **99-66-1**, Valproic acidRL: **Biol (Biological study)**

(peripheral serotonin transporter response to)

RN 99-66-1 HCPLUS

CN Pentanoic acid, 2-propyl- (CA INDEX NAME)



L45 ANSWER 220 OF 220 HCPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1992:626232 HCPLUS Full-text

DOCUMENT NUMBER: 117:226232

TITLE: Effects of oxiracetam on neurotransmitter release from rat hippocampus slices and synaptosomes

AUTHOR(S): Raiteri, M.; Costa, R.; Marchi, M.

CORPORATE SOURCE: Ist. Farmacol. Farmacogn., Univ. Genova, Genoa, 16148, Italy

SOURCE: Neuroscience Letters (1992), 145(1), 109-13

CODEN: NELED5; ISSN: 0304-3940

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of the nootropic drug oxiracetam on the K+-evoked overflow of [³H]D-aspartic acid ([³H]D-ASP), [³H]acetylcholine ([³H]ACh), [³H] γ -aminobutyric acid ([³H]GABA), [³H]noradrenaline ([³H]NA) and [³H]5-hydroxytryptamine ([³H]5-HT) have been studied in superfused rat hippocampal slices. The overflow of [³H]D-ASP was enhanced by low concns. of oxiracetam (0.01-1 μ M) but not by high concns. (10-100 μ M) which showed some tendency to inhibit it. Similarly, low concns. of oxiracetam increased, although less effectively, the depolarization-evoked overflow of [³H]ACh, whereas higher concns. were without effect. At the concns. active on [³H]D-ASP and [³H]ACh overflow oxiracetam did not affect that of [³H]GABA, [³H]NA or [³H]5-HT. The oxiracetam effects present in slices could not be observed in hippocampal synaptosomes. Thus oxiracetam may selectively increase the release of

glutamate and acetylcholine in hippocampus by a mechanism which appears not to be sited in the releasing nerve terminals.

CC 1-11 (Pharmacology)

IT **Nootropics**

(oxiracetam, neurotransmitter release from hippocampus and synaptosomes response to)

IT 50-67-9, 5-Hydroxytryptamine, biological studies 51-41-2, Noradrenaline
51-84-3, Acetylcholine, biological studies **56-12-2**, GABA,
biological studies **1783-96-6**, D-Aspartic acid

RL: **Biol (Biological study)**

(oxiracetam effects on release of, from hippocampus and synaptosomes)

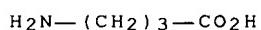
IT **56-12-2**, GABA, biological studies **1783-96-6**, D-Aspartic acid

RL: **Biol (Biological study)**

(oxiracetam effects on release of, from hippocampus and synaptosomes)

RN 56-12-2 HCPLUS

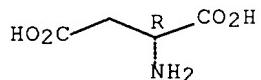
CN Butanoic acid, 4-amino- (CA INDEX NAME)



RN 1783-96-6 HCPLUS

CN D-Aspartic acid (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



INVENTOR NAME SEARCH

=> fil hcap medline embase biosis wpix
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=> s gallagher m/au or gallagher m ?/au or gallagher michela?/au
L46 2406 GALLAGHER M/AU OR GALLAGHER M ?/AU OR GALLAGHER MICHELA?/AU

=> s lund p/au or lund p k?/au or lund pauline/au or lund pauline k?/au
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L48 908 ROTHSTEIN J/AU OR ROTHSTEIN J D?/AU OR ROTHSTEIN JEFF?/AU

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L50 16 DUP REM L49 (20 DUPLICATES REMOVED)
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ANSWERS '9-10' FROM FILE MEDLINE
ANSWERS '11-16' FROM FILE BIOSIS

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L50 ANSWER 1 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2006:333583 HCAPLUS Full-text
DOCUMENT NUMBER: 144:343627
TITLE: Treatment for age-related cognitive decline and other
conditions
INVENTOR(S): **Gallagher, Michela; Lund, Pauline
Kay**
PATENT ASSIGNEE(S): The Johns Hopkins University, USA; University of North
Carolina
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2006034485	A2	20060330	WO 2005-US34331	20050921
WO 2006034485	A3	20061116		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

PRIORITY APPLN. INFO.: US 2004-611749P P 20040921

AB Methods and compns. are disclosed for identifying agents useful for promoting or preserving cognitive function, or for ameliorating cognitive decline, in a mammal. Such agents are identified by screening candidate compds. for an agent that modulates the expression of a gene encoding lipoprotein lipase (LPL), or that modulates the activity of an LPL protein encoded by said gene.

L50 ANSWER 2 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2005:367571 HCPLUS Full-text

DOCUMENT NUMBER: 143:1079

TITLE: GABAB receptor antagonist SGS742 improves spatial memory and reduces protein binding to the cAMP response element (CRE) in the hippocampus

AUTHOR(S): Helm, K. A.; Haberman, R. P.; Dean, S. L.; Hoyt, E. C.; Melcher, T.; *Lund, P. K.*; *Gallagher, M.*

CORPORATE SOURCE: Department of Psychological and Brain Sciences, Johns Hopkins University, Baltimore, MD, 21218, USA

SOURCE: Neuropharmacology (2005), 48(7), 956-964
CODEN: NEPHBW; ISSN: 0028-3908

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Memory storage in the brain requires protein synthesis initiated through signaling pathways that control transcription. Such mechanisms are under active investigation for therapies in disorders involving cognitive dysfunction. Long-term memory can be improved by inhibiting activation or reducing expression of transcription factors such as ATF4/CREB2 and some C/EBP family members which appear to serve as memory suppressors. Here, the authors provide evidence that GABAB receptor antagonists may enhance cognition, at least in part, by this mechanism. The authors tested a GABAB receptor antagonist, SGS742 (CGP36742), on hippocampal-dependent memory and hippocampal nuclear CRE-binding activity in rats. As a result, acute *in vivo* administration of SGS742 both improved memory and reduced total hippocampal CRE-binding activity of which a large proportion in the basal state could be immunoneutralized with CREB2 antibodies. Consistent with its activity on information storage mechanisms, acute SGS742 effectively improved long-term memory in retrograde protocols, in which drug was given at times when memory formation can be interrupted by blocking new protein production. In conclusion, GABAB antagonists may provide a pharmacol. therapy for cognitive impairment, sharing mechanistic features with genetic approaches to reduce CREB2 activity and to augment long-term memory.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L50 ANSWER 3 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3

ACCESSION NUMBER: 2004:467995 HCPLUS Full-text

DOCUMENT NUMBER: 141:34605

TITLE:

Identification of mammalian genes involved in glutamate transport and associated with cognitive function using gene expression profiling of neural tissues, screening of compounds which affect them, drug manufacture and therapeutic uses

INVENTOR(S):

*Gallagher, Michela; Lund, Pauline**Kay, Rothstein, Jeffrey D.*

PATENT ASSIGNEE(S):

Johns Hopkins University School of Medicine, USA

SOURCE:

PCT Int. Appl., 127 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004048551	A2	20040610	WO 2003-US38191	20031124
WO 2004048551	A3	20040812		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2506194	A1	20040610	CA 2003-2506194	20031124
AU 2003302472	A1	20040618	AU 2003-302472	20031124
EP 1567674	A2	20050831	EP 2003-808425	20031124
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003015689	A	20051018	BR 2003-15689	20031124
JP 2006510357	T	20060330	JP 2004-555830	20031124
CN 1829804	A	20060906	CN 2003-80109146	20031124
PRIORITY APPLN. INFO.:			US 2002-428229P	P 20021122
			WO 2003-US38191	W 20031124

OTHER SOURCE(S): MARPAT 141:34605

AB The invention relates to methods of identifying genes involved in cognitive impairment using gene expression profiling of neural tissues, screening of compds. for treating cognitive impairment, and the manufacture and therapeutic use of medicaments to treat cognitive impairment. Impaired cognitive function included mild cognitive impairment, age-related cognitive decline, memory loss, senility, dementia and Alzheimer's Disease. Genes associated with cognitive function included genes involved in glutamate transport. Medicaments were selected for their ability to promote spatial memory acquisition, longterm spatial memory and spatial memory retrieval. An example examines the effects of ceftriaxone treatment on hippocampal gene expression and age-related cognition as measured by the Morris water maze procedure.

L50 ANSWER 4 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4

ACCESSION NUMBER: 2004:1046682 HCPLUS Full-text

DOCUMENT NUMBER: 142:407998

TITLE: Transcriptional mechanisms of hippocampal aging
 AUTHOR(S): *Lund, P. Kay; Hoyt, Eileen C.; Bizon, Jennifer; Smith, Dani R.; Haberman, Rebecca; Helm, Kassie; Gallagher, Michela*
 CORPORATE SOURCE: Department of Cell and Molecular Physiology, University of North Carolina, Chapel Hill, NC, 27599-7545, USA
 SOURCE: Experimental Gerontology (2004), 39(11-12), 1613-1622
 CODEN: EXGEAB; ISSN: 0531-5565

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Aging related cognitive decline is an increasing health problem but affects only a subset of elderly humans. This research uses outbred young (Y) and aged rats. Behavioral characterization distinguishes aged rats with impaired spatial learning (AI) and aged rats with unimpaired learning ability (AU), mimicking the varied susceptibility of the human population to age-associated learning impairment. Studies are testing a hypothesis that hippocampal transcriptional mechanisms and gene expression profiles linked to activator protein-1 (AP-1) and glucocorticoid receptor (GR), mineralocorticoid receptor (MR) or cAMP response element binding protein (CREB) families of transcription factors distinguish successful or unsuccessful aging and cognition. Results from mRNA assays, in situ hybridization, electromobility shift assays and western immunoblot indicate changes in GR and CREB in AI rats. State of the art future approaches to define downstream transcription targets are described.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L50 ANSWER 5 OF 16 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 7

ACCESSION NUMBER: 2001:827660 HCAPLUS Full-text

DOCUMENT NUMBER: 136:83333

TITLE: Effect of age and cognitive status on basal level AP-1 activity in rat hippocampus

AUTHOR(S): Smith, D. R.; Hoyt, E. C.; *Gallagher, M.*; Schwabe, R. F.; *Lund, P. K.*

CORPORATE SOURCE: Department of Psychology, University of North Carolina, Chapel Hill, NC, 27599, USA

SOURCE: Neurobiology of Aging (2001), 22(5), 773-786
 CODEN: NEAGDO; ISSN: 0197-4580

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Activator protein-1 (AP-1) was examined at multiple levels (mRNA, DNA binding, composition) in the hippocampus of young and aged rats that were behaviorally characterized for spatial memory. Glial fibrillary acidic protein (GFAP) mRNA was measured as a gene product known to increase with aging and to be regulated by AP-1. The activity of Jun-N-terminal-kinase (JNK) was also assessed. Levels of c-jun and c-fos mRNAs were unchanged with aging or spatial learning ability. The abundance of GFAP mRNA was significantly increased in aged hippocampus, but did not correlate with spatial learning. Total AP-1 binding activity was unaltered with age or cognitive ability. In the hippocampus of young, aged unimpaired, and aged impaired rats, AP-1 consisted mainly of c-Jun, phosphorylated c-Jun (p-c-Jun), JunD, and smaller amts. of c-Fos. JNK was constitutively active in young and aged hippocampus. It was concluded that the basal expression of c-fos and c-jun mRNA, overall AP-1 binding activity, and AP-1 composition are not influenced by aging or cognitive ability.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L50 ANSWER 6 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 8

ACCESSION NUMBER: 1996:254717 HCPLUS Full-text

DOCUMENT NUMBER: 124:280113

TITLE: Increased expression of type 1 insulin-like growth factor receptor messenger RNA in rat hippocampal formation is associated with aging and behavioral impairment

AUTHOR(S): Stenvers, K. L.; **Lund, P. K.**
Gallagher, M.

CORPORATE SOURCE: Curriculum Neurobiology, University North Carolina, Chapel Hill, NC, 27599, USA

SOURCE: Neuroscience (Oxford) (1996), 72(2), 505-18
CODEN: NRSCDN; ISSN: 0306-4522

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Insulin-like growth factor mRNAs are expressed in adult rat brain. However, little is known about the effects of aging on the expression of the insulin-like growth factors, their receptors, and their binding proteins in different regions of rat brain. The goal of the current study was to assess whether there is altered expression of the insulin-like growth factor system during normal aging in the hippocampal formation, a region particularly vulnerable to the aging process. A spatial learning task in the Morris water maze was used to assess the cognitive status of young (7-8-mo-old) and aged (28-29-mo-old) male Long-Evans rats. Sites of expression and abundance of insulin-like growth factor-I, type 1 insulin-like growth factor receptor, and insulin-like growth factor binding protein-4 mRNAs were then examined by *in situ* hybridization histochem. and solution or northern blot hybridization assays. *In situ* hybridization histochem. revealed no qual. differences in the regional distribution of insulin-like growth factor-I, type 1 receptor, and insulin-like growth factor binding protein-4 mRNAs within the hippocampal formation of young and aged rats. However, quant. anal. of mRNA abundance in hippocampal tissue homogenates showed a significant age-related increase in type 1 receptor mRNA ($t = -2.5$). Furthermore, linear regression anal. indicated that type 1 receptor mRNA abundance was significantly correlated with spatial learning impairment in the water maze ($r = 0.44$) such that greater behavioral impairment was associated with higher type 1 receptor mRNA levels in the hippocampal formation. Neither insulin-like growth factor-I nor insulin-like growth factor binding protein-4 mRNA abundance was related to age or behavior. However, linear regression revealed a neg. correlation between insulin-like growth factor-I mRNA abundance and type 1 receptor mRNA abundance in aged hippocampus ($r = -0.72$). These data indicate that increased hippocampal expression of type 1 receptor mRNA is associated with aging and cognitive decline. The correlation between type 1 receptor and insulin-like growth factor-I mRNA abundance in the hippocampal formation of aged rats suggests that insulin-like growth factor availability may influence type 1 receptor expression. However, because no overall age difference was found in the amount of insulin-like growth factor-I mRNA in the hippocampal formation, decreased insulin-like growth factor from other sources such as the cerebrospinal fluid and the peripheral circulation may be involved in up-regulating type 1 receptor mRNA. Alternatively, type 1 receptor mRNA regulation may be part of a trophic response to the degenerative and regenerative events that occur within the hippocampal formation during aging.

L50 ANSWER 7 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 9

ACCESSION NUMBER: 1994:236962 HCPLUS Full-text

DOCUMENT NUMBER: 120:236962

TITLE: Expression of insulin-like growth factor binding

AUTHOR(S): protein-4 and -5 mRNAs in adult rat forebrain
 Stenvers, Kaye L.; Zimmermann, Ellen M.;
Gallagher, Michela; Lund, P. Kay

CORPORATE SOURCE: Curric. Neurobiol., Univ. North Carolina, Chapel Hill,
 NC, 27599-7320, USA

SOURCE: Journal of Comparative Neurology (1994), 339(1),
 91-105

CODEN: JCNEAM; ISSN: 0021-9967

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Accumulating evidence indicates that the insulin-like growth factors (IGFs) can act as neurotrophic factors. A family of at least six IGF binding proteins (IGFBPs) has been characterized. The IGFBPs prolong the half-life of IGFs in plasma and may modulate IGF action in a cell- or tissue-specific fashion. Two recently characterized IGFBPs, IGFBP-4 and -5, have been shown by northern blot hybridization to be expressed in rat brain, but their cellular sites of synthesis are poorly characterized. Because IGFBP-4 and IGFBP-5 could potentially modulate IGF actions in the brain, the authors used *in situ* hybridization histochem. and 35S-labeled IGFBP-4 and IGFBP-5 riboprobes to localize sites of IGFBP-4 and -5 mRNA expression in adult rat brain. The two IGFBP mRNAs are abundantly expressed within discrete regions of brain. The expression patterns of the two genes are largely nonoverlapping. Notably, IGFBP-4 mRNA is highly expressed within hippocampal and cortical areas, whereas IGFBP-5 mRNA is not detected above background in these areas. Within the hippocampus, abundant IGFBP-4 mRNA expression is detected in pyramidal neurons of the subfields of Ammon's horn and the subiculum and in the granule cell layer of the anterior hippocampal continuation. In the cortex, IGFBP-4 mRNA is widely expressed in most areas and layers. In contrast, IGFBP-5, but not IGFBP-4, mRNA is detected within thalamic nuclei, leptomeninges, and perivascular sheaths. The distinct expression patterns of IGFBP-4 and -5 mRNAs within the brain suggest that these IGFBPs may modulate paracrine/autocrine actions of the IGFs in discrete brain regions or compartmentalization of the IGFs within the brain.

L50 ANSWER 8 OF 16 HCPLUS COPYRIGHT 2007 ACS on STN
 ACCESSION NUMBER: 2006:54235 HCPLUS Full-text
 DOCUMENT NUMBER: 144:121832
 TITLE: Prevention and treatment of cognitive impairment using
 (R)-(-)-5-methyl-1-nicotinoyl-2-pyrazoline (MNP) and
 analogs thereof
 INVENTOR(S): **Gallagher, Michela; Lund, Pauline**
Kay; Selcher, Joel C.; Melcher, Thorsten
 PATENT ASSIGNEE(S): The Johns Hopkins University, USA
 SOURCE: U.S. Pat. Appl. Publ., 16 pp., Cont.-in-part of U.S.
 Ser. No. 722,357.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006014801	A1	20060119	US 2005-58734	20050214
US 2004191803	A1	20040930	US 2003-722357	20031124
PRIORITY APPLN. INFO.:			US 2002-428229P	P 20021122
			US 2003-722357	A2 20031124

OTHER SOURCE(S): MARPAT 144:121832

AB The invention provides methods for improving cognitive function in a subject by administering (R)-(-)-5-methyl-1-nicotinoyl-2-pyrazoline (MNP) or an analog to a subject in need of such treatment. The invention is useful for treatment of cognitive impairment such as mild cognitive impairment (MCI) as well as other conditions.

L50 ANSWER 9 OF 16 MEDLINE on STN DUPLICATE 5
 ACCESSION NUMBER: 2003036405 MEDLINE Full-text
 DOCUMENT NUMBER: PubMed ID: 12543263
 TITLE: Effects of aging on the hippocampal formation in a naturally occurring animal model of mild cognitive impairment.
 AUTHOR: **Gallagher Michela**; Bizon Jennifer L; Hoyt Eileen C; Helm Katherine A; **Lund Pauline K**
 CORPORATE SOURCE: Department of Psychology and Brain Sciences, Johns Hopkins University, 102 Ames Hall, 3400 North Charles St., Baltimore, MD 21218, USA.. michela@jhu.edu
 SOURCE: Experimental gerontology, (2003 Jan-Feb) Vol. 38, No. 1-2, pp. 71-7. Ref: 43
 Journal code: 0047061. ISSN: 0531-5565.
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200305
 ENTRY DATE: Entered STN: 25 Jan 2003
 Last Updated on STN: 13 May 2003
 Entered Medline: 12 May 2003

AB Vertical integration is being used to great advantage in neurobiological research on the basis of age-related cognitive decline. Such research bridges analysis between the molecular and cellular levels and the outcome of impaired psychological functions. Current use of animals models within this paradigm has defined mild cognitive impairment in a subpopulation of outbred aged Long-Evans rats by assessment of hippocampal-dependent spatial cognition. Aged rats with cognitive impairment exhibited no loss of neurons in the hippocampus. Current research is focused on the functional alterations in neurons by methods which assess transcriptional mechanisms and signaling pathways.

L50 ANSWER 10 OF 16 MEDLINE on STN DUPLICATE 6
 ACCESSION NUMBER: 2002138024 MEDLINE Full-text
 DOCUMENT NUMBER: PubMed ID: 11860468
 TITLE: Hypothalamic-pituitary-adrenal axis function and corticosterone receptor expression in behaviourally characterized young and aged Long-Evans rats.
 AUTHOR: Bizon J L; Helm K A; Han J S; Chun H J; Pucilowska J; **Lund P K**; **Gallagher M**
 CORPORATE SOURCE: Department of Psychology, Johns Hopkins University, 3400 North Charles St., Baltimore, MD 21218, USA.. jbizon@jhu.edu
 CONTRACT NUMBER: P01 AG 09973 (NIA)
 SOURCE: The European journal of neuroscience, (2001 Nov) Vol. 14, No. 10, pp. 1739-51.
 Journal code: 8918110. ISSN: 0953-816X.
 PUB. COUNTRY: France
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200204
 ENTRY DATE: Entered STN: 5 Mar 2002
 Last Updated on STN: 24 Apr 2002
 Entered Medline: 23 Apr 2002

AB In the current investigation, hypothalamic-pituitary-adrenal (HPA) axis function was examined in young and aged male Long-Evans rats that were initially assessed on a version of the Morris water maze sensitive to cognitive impairment during ageing. In behaviourally characterized rats, a 1-h restraint stress paradigm revealed that plasma corticosterone concentrations in aged cognitively impaired rats took significantly longer to return to baseline following the stressor than did those in young or aged cognitively unimpaired rats. No differences in basal or peak plasma corticosterone concentrations, however, were observed between young or aged rats, irrespective of cognitive status. Using ribonuclease protection assays and in situ hybridization, we evaluated mineralocorticoid receptor (MR) and glucocorticoid receptor (GR) mRNA abundance in young and aged rats characterized on the spatial task. Abundance of MR mRNA was decreased as a function of age in stratum granulosum but not hippocampus proper, and the decrease in MR mRNA was largely unrelated to cognitive status. However, GR mRNA was significantly reduced in several hippocampal subfields (i.e. stratum granulosum and temporal hippocampus proper) and other related cortical structures (medial prefrontal and olfactory regions) of aged cognitively impaired rats compared to either young or aged cognitively unimpaired cohorts, and was significantly correlated with spatial learning ability among the aged rats in each of these brain regions. In agreement with previous stereological data from this ageing model, no changes were detected in neuron density in the hippocampus of the rats used in the in situ hybridization analysis. These data are the first to describe a coordinated decrease in GR mRNA in a functional brain system including hippocampus and related cortical areas that occurs in tandem with impairments of the HPA response to stress and cognitive decline in ageing.

L50 ANSWER 11 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:198652 BIOSIS Full-text
 DOCUMENT NUMBER: PREV200400199211
 TITLE: Altered nuclear glucocorticoid response element (GRE)
 binding activity and composition in aged hippocampus and
 prefrontal cortex.
 AUTHOR(S): Helm, K. A.; Hoyt, E. C. [Reprint Author]; **Gallagher, M.**; Smith, D. R.; **Lund, P. K.** [Reprint Author]
 CORPORATE SOURCE: Cell. and Mol. Physiology, Univ. North Carolina, Chapel Hill, NC, USA
 SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 397.3.
<http://sfn.scholarone.com>. e-file.
 Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003.
 Society of Neuroscience.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 14 Apr 2004
 Last Updated on STN: 14 Apr 2004

AB Dysregulation of the hypothalamic-pituitary-adrenocortical axis is a hallmark of aging linked to cognitive decline. Excessive glucocorticoid receptor (GR)

activation or loss of GR bearing neurons in the hippocampus and prefrontal cortex (PFC) have been postulated as possible mechanisms but direct analyses of GR activation in age-related cognitive decline is lacking. This study used electromobility shift and supershift assays to examine whether aged rats with impaired spatial learning (AI) have altered levels or composition of activated nuclear transcription factors that bind to a glucocorticoid response element (GRE) in hippocampus or PFC relative to young (Y) or aged unimpaired (AU) rats. Results revealed increases in basal GRE binding activity in hippocampus and PFC of AU and AI relative to Y rats. Supershifts indicated that both corticosteroid (CORT) receptors are components of this binding. Intriguingly, supershifts with p50 or p65 subunits of NFkB indicated these subunits were also components of GRE binding activity. Less hippocampal p50 was associated GRE binding in AI versus Y or AU while p65 did not differ significantly among groups. p65 and p50 were also components of GRE binding in PFC but amounts did not differ across condition. We conclude that aging is associated with increased activation of GRE in hippocampus and PFC independent of increases in nuclear GR or circulating CORT. Age-related increases in nuclear GRE do not correlate with impaired learning but AI rats had reduced levels of NFkB p50 subunit associated with hippocampal GRE. As NFkB has neuroprotective effects and can attenuate the transcriptional activity of GR, our findings suggest that antagonistic effects of NFkB on GR transcription may serve to maintain normal hippocampal function.

L50 ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:472116 BIOSIS Full-text
 DOCUMENT NUMBER: PREV200100472116
 TITLE: Aging involves major increases in hippocampal nuclear binding activity to a glucocorticoid response element (GRE).
 AUTHOR(S): Hoyt, E. C. [Reprint author]; **Gallagher, M.**; Smith, D. R.; Herman, J. P.; **Lund, P. K.** [Reprint author]
 CORPORATE SOURCE: Cell and Molecular Physiology, Univ of North Carolina, Chapel Hill, NC, USA
 SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 232. print.
 Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
 ISSN: 0190-5295.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 10 Oct 2001
 Last Updated on STN: 23 Feb 2002
 AB Glucocorticoids are implicated in age induced cognitive decline and functional degeneration of hippocampal neurons. Our prior studies in Long-Evans rats characterized for spatial learning revealed that aged impaired rats (AI) show no net neuron loss in hippocampus, prolonged stress induced elevation of plasma corticosterone, and a region specific decline in expression of hippocampal glucocorticoid receptor (GR) mRNA relative to aged unimpaired (AU) or young (Y) rats. As a more functional readout of activated GR, this study quantified the levels of hippocampal nuclear proteins which bind GRE in behaviorally characterized Y, AU and AI rats using electromobility shift assays. Significant three fold increases in hippocampal nuclear GRE binding activity were observed in AU or AI relative to Y rats ($p<0.05$) but levels of GRE binding in individual Y or aged rats did not correlate with learning index ($r=0.209$; $p=0.84$). Thus, hippocampal aging involves major increases in

activated transcription complexes that bind GRE but this does not correlate with learning impairment. GRE binds both GR and mineralocorticoid receptor (MR) but GR may mediate hippocampal neurodegeneration and MR neuroprotection. Ongoing studies are testing the hypothesis that AI and AU rats differ in the relative levels of GR and MR associated with GRE, or in their interactions with other modulatory transcription factors.

L50 ANSWER 13 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:76627 BIOSIS Full-text
 DOCUMENT NUMBER: PREV200100076627
 TITLE: Age-related cognitive decline is associated with altered composition of the transcription factor AP-1.
 AUTHOR(S): Smith, D. R. [Reprint author]; Hoyt, E. C.; **Lund, P. K.; Gallagher, M.**
 CORPORATE SOURCE: Univ North Carolina, Chapel Hill, NC, USA
 SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-171.8. print.
 Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000.
 Society for Neuroscience.
 ISSN: 0190-5295.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 7 Feb 2001
 Last Updated on STN: 12 Feb 2002

AB The transcription factor activator protein-1 (AP-1) regulates the transcription of a wide range of genes and is known to regulate some genes that have altered expression in aging, such as GFAP. AP-1 can be composed of dimers of the protein Jun or dimers of the proteins Fos and Jun. JUN:JUN homodimers produce relatively low levels of transcription whereas FOS:JUN heterodimers produce a much higher level of transcription. It is possible that alterations in the relative abundance of Fos and Jun in old rats as compared to young provides a basis for altering the transcriptional function of AP-1. Therefore, we tested the hypothesis that aged rats with impairment on a spatial memory task (water maze) sensitive to alterations in the hippocampal system have concomitant alterations in hippocampal expression of the transcription factor Jun relative to Fos when compared with unimpaired aged rats. Our results indicate that while expression of c-jun and c-fos mRNA is unchanged with aging or impairment, impaired aged rats may exhibit altered binding of specific JUN proteins to AP-1 relative young or unimpaired aged rats.

L50 ANSWER 14 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:533067 BIOSIS Full-text
 DOCUMENT NUMBER: PREV199799832270
 TITLE: Glucocorticoid receptor mRNA expression following selective removal of hippocampal cholinergic input in the rat.
 AUTHOR(S): Han, J. S. [Reprint author]; Pucilowska, J.; **Lund, P. K.; Gallagher, M.** [Reprint author]
 CORPORATE SOURCE: Dep. Psychol., Johns Hopkins Univ., Baltimore, MD 21218, USA
 SOURCE: Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 2106.
 Meeting Info.: 27th Annual Meeting of the Society for Neuroscience. New Orleans, Louisiana, USA. October 25-30,

1997.
 DOCUMENT TYPE: ISSN: 0190-5295.
 Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 Conference; (Meeting Poster)

LANGUAGE: English
 ENTRY DATE: Entered STN: 12 Dec 1997
 Last Updated on STN: 12 Dec 1997

L50 ANSWER 15 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
 STN

ACCESSION NUMBER: 1995:428452 BIOSIS Full-text
 DOCUMENT NUMBER: PREV199598442752

TITLE: Increased hippocampal expression of type 1 insulin-like growth factor (IGF) receptor messenger RNA is associated with cognitive decline in aged rats.

AUTHOR(S): Stenvers, K. L.; **Lund, P. K.**; **Gallagher, M.**

CORPORATE SOURCE: Curriculum Neurobiol., Univ. North Carolina, Chapel Hill, NC 27599, USA

SOURCE: Society for Neuroscience Abstracts, (1995) Vol. 21, No. 1-3, pp. 472.
 Meeting Info.: 25th Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 11-16, 1995.
 ISSN: 0190-5295.

DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 Conference; (Meeting Poster)

LANGUAGE: English
 ENTRY DATE: Entered STN: 3 Oct 1995
 Last Updated on STN: 1 Nov 1995

L50 ANSWER 16 OF 16 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
 STN

ACCESSION NUMBER: 1993:152428 BIOSIS Full-text
 DOCUMENT NUMBER: PREV199344071228

TITLE: Expression of insulin-like growth factor binding protein-4 and -5 mRNAs in adult rat brain.

AUTHOR(S): Stenvers, Kaye L. [Reprint author]; **Gallagher, Michela** [Reprint author]; Zimmermann, Ellen M.; **Lund, P. Kay** [Reprint author]

CORPORATE SOURCE: Curriculum Neurobiol., Univ. North Carolina, Chapel Hill, NC 27599, USA

SOURCE: Society for Neuroscience Abstracts, (1992) Vol. 18, No. 1-2, pp. 951.
 Meeting Info.: 22nd Annual Meeting of the Society for Neuroscience. Anaheim, California, USA. October 25-30, 1992.
 ISSN: 0190-5295.

DOCUMENT TYPE: Conference; (Meeting)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 19 Mar 1993
 Last Updated on STN: 19 Mar 1993

SEARCH HISTORY

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E US2003-722357/APPS

L2 2 SEA ABB=ON PLU=ON L1 OR (US2003-722357/AP OR US2003-722357/PR
N)

SEL RN

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702731-40-6/BI OR 702731-41-7/BI OR 702731-42-8/BI OR 702731-43-
9/BI OR 702731-44-0/BI OR 702731-45-1/BI OR 702731-46-2/BI OR
702731-47-3/BI OR 702731-48-4/BI OR 702731-49-5/BI OR 702731-50-
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73384-59-5/BI OR 9000-81-1/BI OR 9000-97-9/BI OR 99-66-1/BI)

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E COGNITION/CT

E E3+ALL

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E COGNITIVE DISORDERS+ALL/CT

L6 4782 SEA ABB=ON PLU=ON COGNITIVE DISORDERS+PFT,NT/CT

E COGNITION ENHANCERS+ALL/CT

L7 4887 SEA ABB=ON PLU=ON COGNITION ENHANCERS+PFT/CT

L*** DEL 0 S S COGNITION+PFT/CT

E COGNITION+ALL/CT

L8 5895 SEA ABB=ON PLU=ON COGNITION+PFT/CT

L9 12821 SEA ABB=ON PLU=ON (L6 OR L7 OR L8)

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FILE 'REGISTRY' ENTERED AT 14:52:11 ON 24 APR 2007

L10 TRA PLU=ON L9 1-4800 RN : 51133 TERMS (TERM LIMIT EXCEEDED)

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FILE 'HCAPLUS' ENTERED AT 14:53:00 ON 24 APR 2007

L11 TRA PLU=ON L9 1-900 RN : 49745 TERMS

FILE 'REGISTRY' ENTERED AT 14:53:28 ON 24 APR 2007

L12 0 SEA ABB=ON PLU=ON L9 AND PY<2003

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L13 5740 SEA ABB=ON PLU=ON L9 AND PY<2003

FILE 'REGISTRY' ENTERED AT 15:01:26 ON 24 APR 2007

FILE 'HCAPLUS' ENTERED AT 15:01:31 ON 24 APR 2007
L14 6530 SEA ABB=ON PLU=ON L9 AND (PY<2003 OR PRY<2003 OR AY<2003)

FILE 'REGISTRY' ENTERED AT 15:01:55 ON 24 APR 2007

FILE 'HCAPLUS' ENTERED AT 15:02:04 ON 24 APR 2007

FILE 'REGISTRY' ENTERED AT 15:02:41 ON 24 APR 2007
L15 TRA PLU=ON L14 1-1000 RN : 51062 TERMS (TERM LIMIT EXCEEDED)

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FILE 'HCAPLUS' ENTERED AT 15:03:02 ON 24 APR 2007
L16 TRA PLU=ON L14 1-500 RN : 43527 TERMS

FILE 'REGISTRY' ENTERED AT 15:03:23 ON 24 APR 2007
L17 43527 SEA ABB=ON PLU=ON L16
D COST

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FILE 'REGISTRY' ENTERED AT 15:14:23 ON 24 APR 2007
L18 TRA PLU=ON L14 501-1500 RN : 50531 TERMS (TERM LIMIT EXCEEDED)

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L19 TRA PLU=ON L14 501-1250 RN : 49030 TERMS

FILE 'REGISTRY' ENTERED AT 15:17:20 ON 24 APR 2007
L20 49030 SEA ABB=ON PLU=ON L19

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D BIB L14 1251

FILE 'REGISTRY' ENTERED AT 15:20:35 ON 24 APR 2007

FILE 'HCAPLUS' ENTERED AT 15:20:52 ON 24 APR 2007
L21 TRA PLU=ON L14 1251-1350 RN : 4753 TERMS

FILE 'REGISTRY' ENTERED AT 15:20:55 ON 24 APR 2007
L22 4753 SEA ABB=ON PLU=ON L21

FILE 'HCAPLUS' ENTERED AT 15:21:38 ON 24 APR 2007
L23 TRA PLU=ON L14 1351-2350 RN : 22203 TERMS

FILE 'REGISTRY' ENTERED AT 15:22:04 ON 24 APR 2007
L24 22203 SEA ABB=ON PLU=ON L23

FILE 'HCAPLUS' ENTERED AT 15:25:17 ON 24 APR 2007
L25 TRA PLU=ON L14 2351-4451 RN : 21584 TERMS

FILE 'REGISTRY' ENTERED AT 15:26:05 ON 24 APR 2007
L26 21584 SEA ABB=ON PLU=ON L25

FILE 'HCAPLUS' ENTERED AT 15:28:11 ON 24 APR 2007
L27 TRA PLU=ON L14 4451-6530 RN : 23061 TERMS

FILE 'REGISTRY' ENTERED AT 15:29:02 ON 24 APR 2007

L28 23061 SEA ABB=ON PLU=ON L27
 L29 145607 SEA ABB=ON PLU=ON L17 OR L20 OR L22 OR L24 OR L26 OR L28
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 L30 STR
 L31 40 SEA SSS SAM L30
 L32 STR L30
 L33 50 SEA SUB=L29 SSS SAM L30
 L34 2835 SEA SUB=L29 SSS FUL L30

FILE 'HCAPLUS' ENTERED AT 15:43:54 ON 24 APR 2007

FILE 'REGISTRY' ENTERED AT 15:44:00 ON 24 APR 2007

L35 2834 SEA ABB=ON PLU=ON L34/COM

FILE 'HCAPLUS' ENTERED AT 15:44:05 ON 24 APR 2007

L36 394862 SEA ABB=ON PLU=ON L35(L)BIOL+NT/RL
 L37 584 SEA ABB=ON PLU=ON L36 AND L14
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 D KWIC HITSTR 10
 L38 369 SEA ABB=ON PLU=ON L7 AND L37
 D HITSTR

FILE 'REGISTRY' ENTERED AT 15:47:21 ON 24 APR 2007

L39 571 SEA ABB=ON PLU=ON L35 AND NC=1

FILE 'HCAPLUS' ENTERED AT 15:47:36 ON 24 APR 2007

L40 687 SEA ABB=ON PLU=ON L39 AND L14
 L41 362994 SEA ABB=ON PLU=ON L39(L)BIOL+NT/RL
 L42 390 SEA ABB=ON PLU=ON L41 AND L14
 L43 396 SEA ABB=ON PLU=ON L41 AND L7
 L44 396 SEA ABB=ON PLU=ON L41 AND L43
 L45 220 SEA ABB=ON PLU=ON L42 AND L43
 D HITSTR
 D HITSTR 1-10

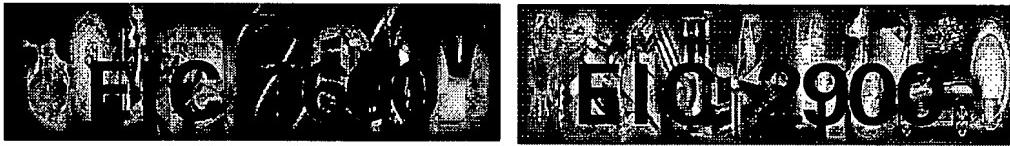
FILE 'HCAPLUS' ENTERED AT 15:51:55 ON 24 APR 2007

D QUE L45
 D L45 IBIB AB HITIND HITSTR 1-10 100-120 200-220

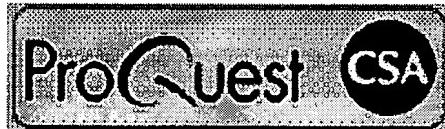
FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS, WPIX' ENTERED AT 15:53:30 ON 24 APR 2007

L46 2406 SEA ABB=ON PLU=ON GALLAGHER M/AU OR GALLAGHER M ?/AU OR
 GALLAGHER MICHELA?/AU
 L47 1274 SEA ABB=ON PLU=ON LUND P/AU OR LUND P K?/AU OR LUND PAULINE/A
 U OR LUND PAULINE K?/AU
 L48 908 SEA ABB=ON PLU=ON ROTHSTEIN J/AU OR ROTHSTEIN J D?/AU OR
 ROTHSTEIN JEFF?/AU
 L49 36 SEA ABB=ON PLU=ON (L46 AND (L47 OR L48)) OR (L47 AND L48)
 L50 16 DUP REM L49 (20 DUPLICATES REMOVED)
 ANSWERS '1-8' FROM FILE HCAPLUS
 ANSWERS '9-10' FROM FILE MEDLINE
 ANSWERS '11-16' FROM FILE BIOSIS
 D L50 IBIB AB TOT

Search Solutions 15-Minute



DEMO @



- Full-text computing journals
- Articles in business, medicine, news, and computer science
- Doctoral dissertations and master's theses
- Full-text journals in education and related fields
- European business and financial information
- Full text of 300+ U.S. and international news sources
 - Telecommunications industry publications

**Library-Main Entrance
Remsen Bldg. Lobby Level
REM 1D 58—Tuesday, April 26th
10:00 or 10:30 AM**

Tuesday, April 24th	Thursday, April 26th
TC 3700 RND 8B31 9:00 AM or 9:30 AM	TC 1600/2900 REM 1D58 10:00 AM or 10:30 AM
TC 2100 RND 4B28 10:30 AM or 11:00 AM	TC 2800 JEF 4B68 11:00 AM or 11:30 AM
TC 3600 KNX 4B68 1:00 PM or 1:30 PM	TC 1700 REM 4B28 1:00 PM or 1:30 PM
TC 2600 KNX 8B59 2:30 PM or 3:00 PM	



No other time will be granted for attendance.

Demo schedules are subject to change.